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THE EVOLUTION OF POSTREPRODUCTIVE LIFE SPAN AS AN INSURANCE AGAINST INDETERMINACY

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Postreproductive life span remains a puzzle for evolutionary biologists. The explanation of increased inclusive fitness through parental care after reproduction that applies for humans is unrealistic for many species. We propose a new selective mechanism, independent of parental care, which relies on the hypothesis that postreproductive life span can evolve as an insurance against indeterminacy: longer life expectancy reduces the risk of dying by chance before the cessation of reproductive activity. We demonstrate numerically that the duration of evolved postreproductive life span is indeed expected to increase with variability in life span duration. An unprecedented assay of 11 strains of the collembola *Folsomia candida* shows the existence of (1) postreproductive life span in the absence of parental care; (2) genetic variability in mean postreproductive life span and postreproductive life span variability itself; (3) strong genetic correlation between latter traits. This new explanation brings along the novel idea that loose canalization of a trait (here, somatic life span) can itself act as a selective pressure on other traits.

KEY WORDS: **Canalization, developmental variability, insurance, longevity, menopause.**

Life-history theory predicts that selection for living beyond the end of reproductive activity is either weak or null (Medawar 1952; Williams 1957). Although for many species, no evidence of postreproductive life span has been documented, survival after the end of reproduction is not unusual in nature. Humans are known for their unusually prolonged postreproductive life span (Austad 1997; Cohen 2004). But menopause and postreproductive life span are not unique to humans and have been observed in the wild and in captivity (Austad 1994, 1997): some nonhuman primates exhibit menopause (Hodgen et al. 1977; Pavelka and Fedigan 1991) and postreproductive life span has also been found in other mammals (Marsh and Kasuya 1984, 1986; Cohen 2004). In those species, postreproductive life span is usually relatively shorter than in humans but, in some cases such as in the killer whale, it can be longer than in humans (Foote 2008; Ward et al. 2009). Postreproductive life span has also been documented in few species with short parental care such as birds raised in captivity (Holmes and Ottinger 2003), or even with no parental care such as nematodes (Klass 1977), guppies (Reznick et al. 2006), or collembola. Two questions arise: (1) why menopause? and (2) given menopause, why live past it? First, menopause—the cessation of the ability to reproduce—might have been selected for instance in humans to protect women from the risk of giving birth after a certain age (Williams 1957; Rogers 1993; Peccei 1995). In its large sense, it can result from the senescence of the reproductive functions such as ovarian exhaustion (Wu et al. 2005; Rashidi and Shanley 2009). In this article, we assume that reproductive cessation exists and we are only addressing the second question.

Given reproductive cessation, several evolutionary forces can select for a prolonged postreproductive life span. In species with strong social structure such as humans, mothers can help their daughters raise their own children during their postreproductive life span. By providing care to their kin, menopaused mothers and grandmothers can thus increase their inclusive fitness (Rogers 1993; Hawkes et al. 1998; Shanley and Kirkwood 2001; Hawkes 2003; Lahdenperä et al. 2004; Shanley et al. 2007; Sear and Mace 2008).

In those species with no direct parental care but with postreproductive life span, various explanations have been given for gains in inclusive fitness, including benefits of grouping, spiteful behavior against nonrelatives, improving foraging efficiency or serving as targets for predators (Bourke 2007). But few explanations have been proposed outside the inclusive fitness argument that is unlikely to apply generally outside some particular species with social behavior. Such explanations include for instance the fact that indirect selection for postmenopausal survival in female human may follow selection for late-survival in males (Tuljapurkar et al. 2007). Finally, other approaches based on optimization models have shown that pleiotropic mutations could also explain moderate late life mortality rates and then some postreproductive life span (Charlesworth 2001; Novoseltsev et al. 2002). Here, we aim to propose conditions where a prolonged postreproductive life span can evolve in species with no parental or grandparental care, and more generally without calling upon inclusive fitness arguments. We propose the hypothesis that postreproductive life span can evolve as an insurance against life span indeterminacy. We derive clear verbal predictions from this hypothesis and support them with a simple mathematical model (described in Supporting information). As a last step, we confront those predictions to an empirical case study of the collembola *Folsomia candida*, a small wingless arthropod featuring no known parental care but with prolonged postreproductive life span. This kind of organism is particularly suitable for addressing this question because clonal reproduction allows direct measurement of genetic differences in mean postreproductive life span and in the level of life span indeterminacy. Our data show that these two traits are, as we predict, strongly genetically correlated.

Starting from sexual maturity, let us first define two types of life spans: the "reproductive life span" (gray segment in Fig. 1) during which an individual is able to reproduce and the "somatic life span" that terminates at death. An individual will enjoy a "postreproductive life span" (black end arrow in Fig. 1) if its somatic life span is longer than its reproductive life span (Fig. 1, genotype). If one assumes that reproductive life span is fixed, fitness will only depend on the somatic life span, which can vary. The crux here is that the effect of the somatic life span indeterminacy is asymmetrical: the fitness is not modified if the somatic life span is longer than the reproductive life span (Fig. 1, phenotype 1) but it is reduced when the somatic life span is shorter than the potential reproductive life span (then there is no postreproductive life span, Fig. 1 phenotype 2). Hence, if somatic life span varies, it pays to have an average somatic life span larger than reproductive life span so that variation around this mean does not encroach on reproductive life span. Similarly, when reproductive life span varies it pays to hedge by increasing somatic life span beyond the average reproductive life span.

Two predictions can be made from this verbal model:

- When there is no variance in the realization of reproductive and somatic life spans, somatic life span should evolve to a length slightly smaller than reproductive life span, due to mutation–selection balance.
- Postreproductive longevity should evolve as a consequence of variance in reproductive and/or somatic life span. The duration of the evolved mean predicted postreproductive longevity should increase with increasing level of life span variance.

We have verified these predictions in a theoretical model described in the Supporting information as well as through the analysis of an empirical example based on the collembola *F. candida* where postreproductive life span has been observed.

Material and Methods

BIOLOGICAL MODEL

We used 11 clonal strains of the parthenogenetic collembola (commonly called "springtail") *F. candida* (Isotomidae, Willem, 1902) to measure and compare their postreproductive life spans and to study possible association between mean duration of their postreproductive life span and their variability. This species can be easily reared in the laboratory and is commonly used as a standard model for soil arthropods (Fountain and Hopkin 2005). For each of the 11 clones (Tully et al. 2006), 20 individuals were isolated at birth and placed individually in standard rearing boxes under controlled conditions: 21°C and 100% relative humidity (see Tully and Ferrière 2008 for methodological details). To study whether a potential correlation between mean postreproductive longevity and longevity variance was stable across different environments, we raised our collembola under two contrasted food regimes: for each clone, food (dried pellets of agar and yeast) was provided ad libitum for 10 individuals (later referred as the "ad libitum food treatment"), whereas the other 10 individuals had access to food only one day per week (dietary restriction treatment). The rearing boxes were checked at least twice a week. The eggs that were laid were counted at the same time and then removed from the boxes. We define the reproductive life span as age at last egg laying and somatic life span as age at death. These definitions provide a simple, straightforward measurement of postreproductive life

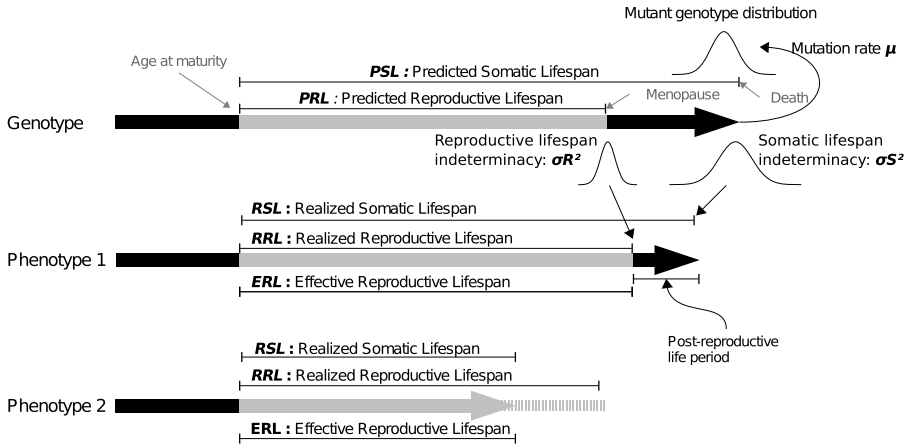

Figure 1. Diagram representing the model and its parameters. Each genotype is defined by a genetically fixed predicted somatic and reproductive life span (*PSL* and *PRL*). Due to some trait indeterminacy (variance on the somatic and reproductive life span), it can produce different phenotypes, which will differ by the realized values of their somatic and reproductive life span (*RSL* and *RRL*). The fitness of each phenotype is a function of its effective reproductive life span (*ERL*) that equals reproductive life span if somatic life span extends beyond reproductive cessation (Phenotype1) or somatic life span if the individual dies before the end of its reproductive life (Phenotype 2).

span (difference between somatic and reproductive life span). We kept in our analyses those individuals that may have died during an interclutch interval that lasts on average 9.3 days under ad libitum food and 18 days under dietary restriction. But overall mean postreproductive life span was longer than these interclutch intervals (26 days and 66 days under ad libitum food and dietary restriction, respectively). During this experiment, eight individuals were accidentally killed and were therefore removed from the analysis.

STATISTICAL ANALYSIS

In our verbal and mathematical models, the somatic life span (S) evolves whereas reproductive life span (R) is supposed to be fixed. In the biological model, reproductive life span varies between individuals, clones, and food regimes (Fig. 2). Therefore, to make any meaningful comparison, one needs to take the genetic and environmental variance of mean reproductive life span into account. We have done it in two ways for both the measurement of mean postreproductive life span and the life span variance.

Mean postreproductive life span

For postreproductive life span, we either used the raw measurements of postreproductive life span (S-R) or used a measurement adjusted for generation time. Using data on survival and reproduction through life, generation time (T) was computed for each clone and environment and used to built up this adjusted measurement, (S-R)/T (see Supporting information). Generation time—the average age of reproduction—has indeed been shown to be a reliable metric to assess the relative importance of life-history variables between populations (Gaillard et al. 2005). In both cases, the measurements of postreproductive life span—computed for each individual—were analyzed with a linear model using generalized least squares (*gls* function from the software R, Ihaka and Gentleman. 1996) with interaction between clone and food ration (high food vs. dietary restriction) as fixed effect. We took into account the heteroscedasticity by using the variance function *varIdent* to model the variance structure between the different clones under each food ration (Pinheiro and Bates 2002).

Life span variance

Life span variance was measured either on the raw data of postreproductive life span (S-R) or on somatic life span adjusted for reproductive life span (S/R). To have an estimate of the life span indeterminacy, we ran another model in which postreproductive life span was analyzed with a linear model (*gls*) with clone × food ration as fixed effect. As before, we used the *varIdent* function to model under each food ration, the clone differences in variance. This variance part of the model was used as a measure of genetic differences in life span indeterminacy.

Genetic correlation

We then studied within each food regime, the genetic correlation between these different measurements of postreproductive life span and of life span variance (Figs. 3B and S2).

Results

THEORETICAL MODEL

When there is no developmental variance on life span, the somatic life span evolves to a mean length slightly smaller than

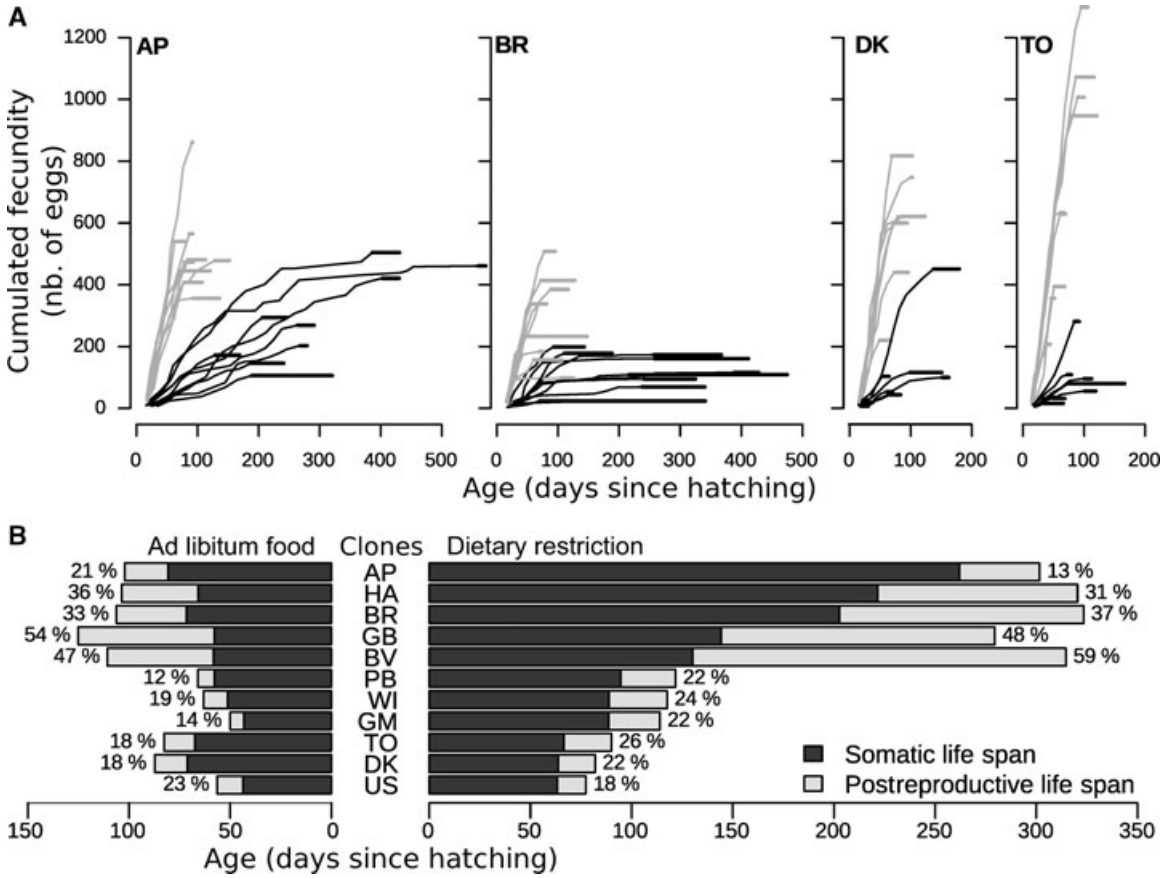

Figure 2. **Reproductive and postreproductive life span in the collembola. (A) To illustrate the diversity of individual and clonal reproductive and postreproductive life span, we have plotted the curves of cumulated fecundity for four clones (AP, BR, DK, and TO). Each curve represents the reproductive trajectory of a single individual from hatching to natural death. The collembola were raised either under ad libitum food regime (gray) or under dietary restriction (black). Postreproductive life spans are highlighted with a bold line. (B) Mean genetic reproductive life span (dark gray) and mean genetic postreproductive life span (light gray) are represented for each clone in the ad libitum food treatment (left) or under dietary restriction (right, 10 individuals per clone per treatment). The percentages express the mean proportion of total life span an individual spends in postreproductive state according to its clone and to the food treatment. It is on average only slightly shorter under ad libitum food ration (24% vs. 28% on average, $F_{1,196} = 4.2$, $P = 0.04$) but varies between clones. Clone labels are the same as in Tully et al. 2006.**

reproductive life span (see Table S1 and Figs. 3A and S1, where mean evolved predicted somatic life span equals 38.8 vs. 40 for reproductive life span). In the presence of developmental variance on life span, a postreproductive life span emerges. The duration of the mean evolved postreproductive life span increases with increasing levels of somatic and reproductive life span variances (Table S1 and Figs. S1 and 3A), both sources of variance having almost additive effects (Fig. 3A, difference between the gray and black line).

BIOLOGICAL MODEL

Most individuals were found to live for quite a long time after laying their last clutch (Fig. 2A): depending on the food regime and the clone identity, springtails lived on average between about 50 days (clone "GM" in high food regime) and about 320 days (clone "BR" under dietary restriction, Fig. 2B). The individuals spent on average between 12% strain ad libitum food ration than under the restricted one (36 vs. 47 days $F_{1,196} = 14.3$, $P < 0.001$, Figs. 2B and S2) but the adjusted postreproductive life span (S-R)/T did not differ between food regimes ($F_{1,196} = 1.41$, $P = 0.23$, Fig. 3B, see also Fig. 2B). These traits varied between clones ($F_{10,196} > 5.7$, $P < 0.001$). We also found a significant difference between clones in the variance of postreproductive life span (S-R) and of relative somatic life span (S/R, $\chi^2_1 > 245$, $P < 0.0001$). These latter effects were used as a surrogate of the genetic somatic life span variances. Finally, and most importantly, we found as predicted that the postreproductive life span, genetic means were positively associated with the genetic levels of postreproductive life span variances. This is the case under both food rations (Figs. 3B and S2).

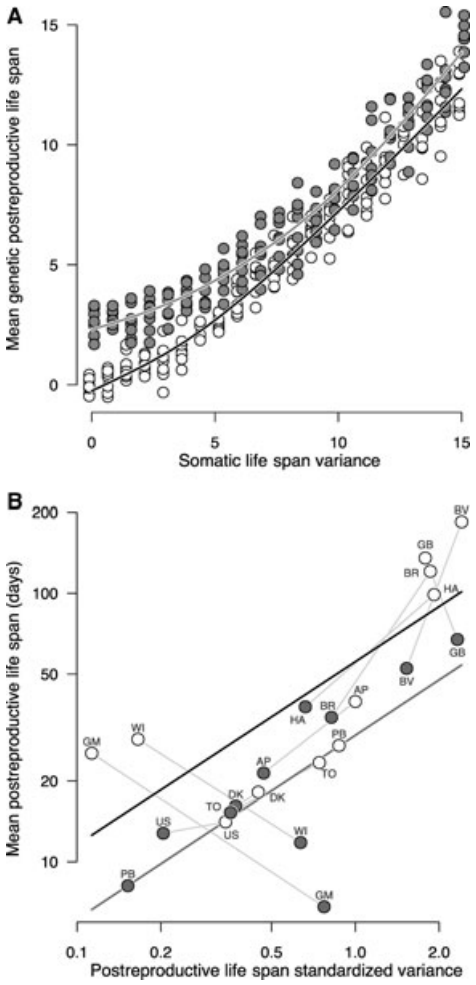

Figure 3. Mean postreproductive life span and life span variance. (A) Mathematical model. Mean evolved postreproductive life spans are plotted as a function of somatic life span developmental variances σS^2 (x-axis) and reproductive life span developmental variance σR^2 (white circles, $\sigma R^2 = 0$; gray circles, $\sigma R^2 = 5$). The black and gray lines are nonlinear functions adjusted to the two groups of data. Each point is the mean evolved life span of a simulated population between 1000 and 3000 generations. (B) Biological model. The mean postreproductive life spans (adjusted for differences in generation time) are plotted against the corresponding variance of relative postreproductive life span for the 11 clones raised under two food treatments (ad libitum, gray disks; dietary restriction, white disks). Each point is the mean genetic values of one of the 11 clones under one of the two food treatments (see Statistical methods). The light gray lines show the bivariate reaction norms. Variances are scaled to the clone AP in low food treatment. The two regression lines show that the traits are genetically correlated in both environments (gray line and filled circles: ad libitum, $R^2 = 0.95$ [0.81–0.98], $t_9 = 9.2$, $P<0.001$; black line and open circle: dietary restriction, $R^2 = 0.90$ [0.64;0.93], $t_9 = 6.1$, $P < 0.001$). The left axis represents the scaled genetic means in each environment for a 60 days long generation (i.e., the overall mean generation time). The right axis express the adjusted postreproductive life span as a percentage of the measured generation time for each clone and food ration.

Discussion

MODEL PREDICTIONS

We verified that indeterminacy on the realization of genetically programmed life span is sufficient to drive the evolution of natural selection will therefore favor individuals with a sufficiently long predicted postreproductive life period that functions as a buffer to developmental variance. The length of the evolved postreproductive life span is proportional to the level of this indeterminacy that can affect either the somatic life span, the reproductive life span or both of them.

In our model, mutation load was the only evolutionary force that tends to decrease postreproductive life span or to slow down its increase. No trade-off was involved between longevity and reproduction based, for instance, on allocation in maintenance when available resources are limited (Flatt and Schmidt 2009). Including such trade-off would have probably shortened the evolved postreproductive life span. But the overall effect of life span indeterminacy on mean postreproductive life span would probably have remained qualitatively the same. Moreover, it has been shown that resource allocation based trade-offs need not to occur systematically (Mukhopadhyay and Tissenbaum 2007) and that, as in our model, the evolution of reproduction and survival can be uncoupled (Barnes and Partridge 2003).

CONFRONTATION WITH THE BIOLOGICAL MODEL

In the real living world, is it possible that postreproductive life span has evolved to provide such an insurance against life span indeterminacy? It is difficult to provide a clear answer to this question.

First, the real cost of life span indeterminacy is probably much smaller in nature than in our model simply because most individuals never live until the end of their potential reproductive life span, due to external causes (e.g., injury, disease, predation). Moreover, reproduction is supposed to terminate abruptly in our model whereas in the real world a progressive—also sometimes quite rapid—reduction of the reproductive performance can usually be observed before an eventual complete cessation (Packer et al. 1998; Holmes et al. 2003; Ricklefs et al. 2003). Most real populations are age-structured and generations are overlapping, which is not the case in our model. Therefore, the model does not take into account the decline of marginal fitness gains of survival with age. Then selective force that drives the evolution of postreproductive life span in our study is probably much weaker for most organisms in nature. More work is needed to study the joint evolution of a progressive reduction of the reproductive activity with the evolution of abruptly terminated somatic life. Using a classical age-structured aging model would enable to take into account a more realistic progressive decrease of fertility and survival over age classes late in life. Such a life table modeling approach would also enable to relax

our model assumptions of asexuality and stable population size.

Second, it is not easy to demonstrate such an effect because one has to measure the intrinsic genetic mean and variance of the reproductive and somatic life span in a constant and controlled experiment. Yet, the collembola provides a good experimental system for such measurements. They first illustrate the possibility of a quite long postreproductive life span (about 25% of the total life span on average) even in species with no known parental care. This first result contradicts the claim that prolonged postreproductive life span is almost unique to humans and quite rare in other species (Pavelka and Fedigan 1991; Hill and Kaplan 1999). In the light of this example, which reinforces other recent observations (Reznick et al. 2006; Ward et al. 2009), we think that postreproductive life span is probably more common than usually expected. Its rarity in the literature may be due to the difficulty of observing patterns of senescence in nature (Nussey et al. 2008) and of conducting long-term longitudinal follow-up of identifiable individuals (Carey 2003; Monaghan et al. 2008).

Our experiment also shows strong genetic differences between our clones in reproductive life span, somatic life span, and also in the level of life span indeterminacy. The clones come from several geographical origins (Tully et al. 2006) where they might have evolved under diverse environments selecting for contrasted reproductive strategies (Tully and Ferrière 2008). Such within-species variability in reproductive and somatic life span has been observed already in natural populations (Fox et al. 2004; Tatar 2001) and on individuals maintained in the laboratory (Carey 2003). These variations can result from genetic differences between individuals and from differences in environmental conditions (Horiuchi 2003). Even when genetic and environmental sources of variation are kept minimal—by raising clonal individuals in the laboratory for example, as in our case study below—the duration of both reproductive and somatic life span can remain largely variable (de Haan et al. 1998; Roark and Bjorndal 2009). This indeterminacy can result from epigenetic effects, from noise in gene expression (instability of developmental processes), from fluctuations in population dynamics, or from micro-environmental noise (DeWitt et al. 1998; Flatt 2005; Fraga 2009; Landry 2009). Dietary restriction was found to extend both reproductive and postreproductive life span in similar proportion, the percentage of time spent in postreproductive state remaining about the same for each clone in the two environments (Fig. 2B).

Finally, and more importantly, the observed genetic diversity is not randomly organized: under both diets, the genetic levels of life span indeterminacy are positively correlated with the genetic levels of mean postreproductive life span (Figs. 3B and S2). This genetic correlation is in a perfect agreement with our model prediction—the higher the life span indeterminacy, the longer the expected postreproductive life span—even though our biological model does not fit all our theoretical model hypotheses. The mean reproductive life span varies substantially within clones, whereas it is supposed to be genetically fixed in our model. One could argue that this correlation reflects a classical correlation between the mean and the variance of a trait without resulting from a specific selective process. But then one would expect to observe a similar correlation between the mean and variance of somatic life span (S), which is not the case: the genetic correlation between mean and variance almost vanishes when one does not correct for variance in reproductive life span (Fig. S3). The genetic correlation indicates that life span indeterminacy and postreproductive life span do at least partly coevolve. Although this correlation comforts the biological validity of our model, it is nevertheless not a real proof of its validity. The causal relationship could be reversed—the life span indeterminacy being a secondary consequence of a prolonged postreproductive life span—or could be due to a third hidden factor. Nevertheless, whatever the direction of the causal relationship, there is a genetic link between life span indeterminacy and mean postreproductive life span.

CONCLUSION

Our framework provides a simple mechanism to explain the evolution of postreproductive life span. This mechanism does not rely on any kind of mother or grandmother helping or on any other kind of inclusive fitness. It can therefore be applied to species with absolutely no parental or grand-parental care. In this framework, postreproductive life span can be understood as a byproduct of the insurance of being able to benefit from one's whole reproductive life span despite individual variability in somatic life span. In a way, selection has favored the evolution of grandmothers with better quality than apparently needed. This compares with the design of airplanes, which are built in such a way to have a mean durability that is longer than their expected service life (Saunders, 1968). This generates a safety period, which reduces the risk of a catastrophic failure during the service life.

Our study brings to the fore the importance of developmental variability as an evolutionary force. This evolutionary force has a very specific feature: as the developmental variance increases, the average effect of selection on the whole population intensifies, but the pressure exerted on individuals gets more variable. A same genotype can produce individuals with long or short life span, the latter only being subject to purifying selection. The intensity of this selection varies within individuals even though they share the same genotype and live in the same environment. This work stresses the importance of sources of phenotypic indeterminacy in shaping life histories. However, the origins and adaptive significances of these sources of variability largely remain to be addressed.

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Supporting Information

The following supporting information is available for this article:

Table S1. Statistical table regarding the effects of somatic and reproductive developmental variances on population mean evolved predicted somatic life spans.

Figure S1. Mean predicted somatic longevity as a function of time.

Figure S2. Mean postreproductive life span and life span variance for the 11 clones raised under two food treatments (ad libitum, gray disks and gray regression line; dietary restriction, white disks and black regression line).

Figure S3. The genetic means and variances of raw values of somatic life span are correlated under dietary restriction (log–log regression $R^2 = 0.71$, $t_9 = 3.0$, $P = 0.01$) but not in ad libitum conditions ($t_9 = 0.33$, $P = 0.74$). See Figure S2 captions for details.

Supporting Information may be found in the online version of this article.

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